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(FILE 'HOME' ENTERED AT 14:05:18 ON 03 AUG 2000)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 14:06:06 ON 03 AUG 2000

L1 10 S HUMAN(5A)(METHIONINE(W)SYNTHASE(W)REDUCTASE OR MTRR)
L2 7 DUP REM L1 (3 DUPLICATES REMOVED)

=> d 1-7 au ti so l2

L2 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2000 ACS
IN Gravel, Roy A.; Rozen, Rima; Leclerc, Daniel; Wilson, Aaron; Rosenblatt, David
TI **Human methionine synthase reductase** : cloning, and methods for evaluating risk of neural tube defects, cardiovascular disease, cancer, and down's syndrome
SO PCT Int. Appl., 85 pp.
CODEN: PIXXD2

L2 ANSWER 2 OF 7 BIOSIS COPYRIGHT 2000 BIOSIS
AU Yi, P.; Hobbs, C.; Melnyk, S.; Sherman, S.; Gravel, R.; Wu, Q.; Rozen, R.; James, S. J.
TI Polymorphisms in the methylenetetrahydrofolate reductase (MTHFR) and in the methionine synthase reductase (MTRR) genes increase maternal risk of Down syndrome.
SO FASEB Journal, (March 15, 2000) Vol. 14, No. 4, pp. A231. print..
Meeting Info.: Annual Meeting of Professional Research Scientists: Experimental Biology 2000. San Diego, California, USA April 15-18, 2000 Federation of American Societies for Experimental Biology
. ISSN: 0892-6638.

L2 ANSWER 3 OF 7 BIOSIS COPYRIGHT 2000 BIOSIS
AU Wilson, Aaron; Platt, Robert; Wu, Qing; Leclerc, Daniel; Christensen, Benedicte; Yang, Hong; Gravel, Roy A.; Rozen, Rima (1)
TI A common variant in methionine synthase reductase combined with low cobalamin (vitamin B12) increases risk for spina bifida.
SO Molecular Genetics and Metabolism, (Aug., 1999) Vol. 67, No. 4, pp. 317-323.
ISSN: 1096-7192.

L2 ANSWER 4 OF 7 BIOSIS COPYRIGHT 2000 BIOSIS
AU Fodinger, Manuela (1); Buchmayer, Heidi; Sunder-Plassmann, Gere
TI Molecular genetics of homocysteine metabolism.
SO Mineral and Electrolyte Metabolism, (July Dec., 1999) Vol. 25, No. 4-6, pp. 269-278.
ISSN: 0378-0392.

L2 ANSWER 5 OF 7 MEDLINE DUPLICATE 1
AU Leclerc D; Odi`evre M; Wu Q; Wilson A; Huizenga J J; Rozen R; Scherer S W; Gravel R A
TI Molecular cloning, expression and physical mapping of the **human methionine synthase reductase** gene.
SO GENE, (1999 Nov 15) 240 (1) 75-88.

Journal code: TCP. ISSN: 0378-1119.

L2 ANSWER 6 OF 7 BIOSIS COPYRIGHT 2000 BIOSIS
AU Brown, Charlotte A. (1); McKinney, Kimberly Q. (1); Kaufman, Jay S. (1); Gravel, Roy A.; Rozen, Rima
TI Association of gene polymorphisms in methylenetetrahydrofolate reductase, methionine synthase and methionine synthase reductase with homocysteine levels and coronary artery disease.
SO Circulation, (Nov. 2, 1999) Vol. 110, No. 18 SUPPL., pp. I.754.
Meeting Info.: 72nd Scientific Sessions of the American Heart Association Atlanta, Georgia, USA November 7-10, 1999
ISSN: 0009-7322.

L2 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2000 ACS
AU Leclerc, D.; Wilson, A.; Dumas, R.; Gafuik, C.; Song, D.; Watkins, D.; Heng, H. H. Q.; Rommens, J. M.; Scherer, S. W.; Rosenblatt, D. S.; Gravel, R. A.
TI Cloning and mapping of a cDNA for methionine synthase reductase, a flavoprotein defective in patients with homocystinuria
SO Proc. Natl. Acad. Sci. U. S. A. (1998), 95(6), 3059-3064
CODEN: PNASA6; ISSN: 0027-8424

=> d ab 6 7 l2

L2 ANSWER 6 OF 7 BIOSIS COPYRIGHT 2000 BIOSIS

L2 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2000 ACS
AB Methionine synthase catalyzes the remethylation of homocysteine to methionine via a reaction in which methylcobalamin serves as an intermediate Me carrier. Over time, the cob(I)alamin cofactor of methionine synthase becomes oxidized to cob(II)alamin rendering the enzyme inactive. Regeneration of functional enzyme requires reductive methylation via a reaction in which S-adenosylmethionine is utilized as a Me donor. Patients of the cblE complementation group of disorders of folate/cobalamin metab. who are defective in reductive activation of methionine synthase exhibit megaloblastic anemia, developmental delay, hyperhomocysteinemia, and hypomethioninemia. Using consensus sequences to predicted binding sites for FMN, FAD, and NADPH, a cDNA corresponding to the "methionine synthase reductase" reducing system required for maintenance of the methionine synthase in a functional state was cloned. The gene MTRR has been localized to chromosome 5p15.2-15.3. A predominant mRNA of 3.6 kb is detected by Northern blot anal. The deduced protein is a novel member of the FNR family of electron transferases, contg. 698 amino acids with a predicted mol. mass of 77,700. It shares 38% identify with human cytochrome P 450 reductase and 43% with the C. elegans putative methionine synthase reductase. The authenticity of the cDNA sequence was confirmed by identification of mutations in cblE patients, including a 4-bp frameshift in two affected siblings and a 3-bp deletion in a third patient. The cloning of the cDNA will permit the diagnostic characterization of cblE patients and investigation of the potential role of polymorphisms of this enzyme as risk factor in hyperhomocysteinemia-linked vascular disease.